CHAPTER 1

Abdominal Pain

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The most widely accepted recommendations for the management of acute abdominal pain in the office and emergency department settings are summarized in a consensus statement from the American College of Emergency Physicians (ACEP), a clinical policy for the initial evaluation and management of patients presenting with a chief complaint of nontraumatic acute abdominal pain. These guidelines recommend against restricting the differential diagnosis solely by the location of pain, as well as not using the presence or absence of a fever to distinguish potential surgical from medical etiologies of abdominal pain. In cases in which the diagnosis is unclear, serial abdominal examinations over several hours or ancillary testing may help improve the diagnostic accuracy in patients with unclear causes of abdominal pain. Stool for occult blood testing, as well as a pelvic examination in females, should be strongly considered in all patients.

The ACEP statement recommends that patients at a high risk for atypical presentations of acute abdominal pain should be identified early in their presentation to avoid potential misdiagnosis. An electrocardiogram (ECG) should be considered in older adult patients, in those with cardiac risk factors, and in diabetic patients with upper abdominal pain of unclear etiology. A pregnancy test should be obtained in women of childbearing potential. The diagnosis of appendicitis should be considered (though testing may not be required) in women with pelvic inflammatory disease or urinary tract infections because occasionally pelvic examination findings and urinalysis may be positive in patients whose pain is caused by an inflamed appendix. Appropriate narcotic analgesia should be provided to patients being evaluated for abdominal pain in the emergency department.

This chapter focuses on several of the most common conditions related to the gastrointestinal (GI) system. Conditions that may cause significant abdominal pain include cardiovascular, gynecologic, pulmonary, renal, and other various etiologies.

GASTROINTESTINAL

APPENDICITIS

Symptoms

- Right lower quadrant (RLQ) migratory abdominal pain +++++
- Fever ++++

- Anorexia +++
- Nausea +++
- Vomiting +++
- Tenesmus ++

Signs

- Abdominal tenderness and guarding (may indicate peritoneal irritation) +++++
- Tenderness at McBurney's point (two thirds of the distance from the umbilicus along a straight line to the anterior superior iliac spine of the pelvis) ++++
- Rovsing's sign (referred tenderness from the left lower quadrant [LLQ] to the RLQ during palpation) +++
- Psoas sign (pain elicited by extending the hip posteriorly with the patient lying prone) ++
- Obturator sign (pain elicited by abducting the right hip with the patient lying supine) ++

Note: No sign or symptom has been shown to be completely reliable in predicting acute appendicitis.

Workup

- The patient may walk in a flexed or guarded fashion, alerting the clinician to consider peritoneal irritation due to ruptured appendix.
- Auscultation of bowel sounds is not considered to be helpful in examining a child for potential appendicitis because they tend to be hypoactive or absent once peritonitis occurs after appendiceal perforation.
- Rectal examination should be performed to rule out anorectal pathology (e.g., abscess).
- In the past, it was thought that pain medications should not be administered prior to surgical evaluation. Recent literature demonstrates the safety of this practice, which may in fact improve the reliability of abdominal examination.
- No laboratory studies have been proven to be adequately predictive of acute appendicitis. Complete blood count with platelets and differential (CBCPD) do not have adequate sensitivity or specificity to be particularly useful. Urinalysis may be helpful, but may be falsely positive or negative.
- A urine pregnancy test should be performed to rule out complications of pregnancy such as ectopic pregnancy in women of childbearing age.
- Recent advances in computed tomography (CT) scan technology and accuracy have led to increased reliance on CT for diagnosis. Diagnostic imaging is not necessarily required when there is either a high or low probability of appendicitis, when a clinical diagnosis can be made.
- Plain film radiographs of the abdomen may reveal a fecalith in the appendix in 10% of cases, but are not routinely ordered because they are almost never diagnostic.
- Ultrasound (US) may be advantageous in thin patients. CT is the preferred choice in obese patients.

- If the abdominal CT does not show evidence of acute appendicitis, the patient may either be admitted for observation or discharged at the discretion of the examiner and/or parents with instructions for follow-up if symptoms worsen.

Comments and Treatment Considerations

In major U.S. hospital centers, the incidence of appendiceal perforation in the general pediatric population ranges from 20% to 40%. These patients are often dehydrated and toxic appearing with obvious physical signs of peritonitis, and should be immediately fluid resuscitated and treated with broad-spectrum antibiotics prior to being taken to the operating room.

There is a small subgroup of patients with appendiceal perforations in whom the diagnosis is missed or who do not present for medical evaluation until late in the course of the illness. In these patients who may have been ill for a period of 7 to 10 days, radiographic studies often reveal a walled-off abscess or phlegmon in the RLQ. These patients can often be treated medically and have their abscesses drained percutaneously by an interventional radiologist, then sent home to convalesce when stable. After 6 weeks, surgeons may opt to perform an interval appendectomy.

After completion of a careful history and physical examination and equivocal laboratory and imaging studies, there are still patients in whom a diagnosis of appendicitis remains unclear. A diagnostic laparoscopy is an acceptable procedure in this group of patients, and during the procedure the appendix should generally be resected, regardless of appearance, and sent to pathology.

Whether an appendectomy should be performed via traditional open laparotomy or via laparoscopy is debatable. Guidelines recommend that the primary modality should be the surgeon's choice because equivalent success and complication risks are seen following both procedures. In obese patients the laparoscopic approach may have an advantage, yielding decreased operating time and a shorter hospital stay.

Preoperative antibiotics should be routinely administered as soon as possible after presentation in all patients with appendicitis. Evidence to support the use of any specific antibiotic or combination of antibiotics is lacking. Broad-spectrum antibiotics may provide effective coverage but may also increase the risk of susceptibility to multidrug-resistant organisms.

Infectious complications related to appendicitis include intra-abdominal abscesses, peritonitis, and wound infection. Rates of postoperative wound infection vary between 6% and 50% based primarily on antibiotic coverage and perforated versus nonperforated appendicitis. Antibiotics should be discontinued once the patient is afebrile, tolerates a regular diet, and demonstrates a normal white blood cell (WBC) count without a leftward shift to band neutrophils.

Feeding the patient after perforated appendicitis may be instituted when any ileus or bowel obstruction secondary to the perforation has resolved as indicated by a flat, soft abdomen and the presence of flatulence.

Education for the patient and family should address the treatment plan, advancement of diet, pain management, antibiotic administration if indicated, incision care, signs and symptoms of infection to watch for, when to return to school or daycare, and the need for follow-up appointments.

CHOLECYSTITIS

See Chapter 28, Jaundice.

Constipation and Fecal Impaction

Constipation is a common symptom with a wide variety of interpretation from patients regarding its etiology and the actual frequency of stooling; it is commonly defined as passing fewer than three stools per week. Many patients attempt to remedy their constipation with over-the-counter laxatives with modest success. For some patients, especially children, constipation and fecal impaction can become a chronic condition that requires a detailed workup to rule out functional and/or systemic disease conditions.

Symptoms

- Diffuse, crampy abdominal pain +++

Signs

- Often physical examination is unremarkable. ++++
- Physical examination may provide clues to systemic disease that contributes to constipation (e.g., hypothyroidism). ++
- Abdominal mass, potentially palpated in a child or thin adult ++

Workup

- Evaluation may include flexible sigmoidoscopy, barium enema, or colonoscopy in patients with protracted constipation that has not resolved spontaneously and in older patients for whom change in bowel habits may require further evaluation to rule out tumor or other pathology.
- Serum electrolytes, thyroid function tests, blood glucose, and serum calcium to evaluate for a metabolic disorder that may cause constipation, but this is a very uncommon cause
- Fecal occult blood testing (FOBT) as a screen for GI lesions
- Anorectal manometry may be indicated to rule out Hirschsprung's disease.
- Rectal biopsy may also be indicated in the workup of Hirschsprung's disease. Full-thickness rectal wall mucosa and muscularis must be obtained to adequately exclude the presence of myenteric ganglia.

Comments and Treatment Considerations

Symptoms may be improved by lifestyle changes, including exercise and allowing adequate time to defecate. Increase dietary fiber

through fruits, vegetables, grains, or supplemental fiber, for example. Increase fluid (water) intake. Avoid medications that may worsen symptoms (e.g., opioids, calcium channel blockers). Liberal laxative use, including mineral oil, lactulose or polyethylene glycol (PEG) may be helpful. Sometimes a vicious cycle develops: constipation is relieved by laxatives or cathartics; the patient has no urge to pass stool for several days because the bowel has been evacuated; the patient has no urge to pass stool for several days and becomes concerned; then the patient perceives constipation again and resumes laxatives. Fecal impaction may be broken manually during digital rectal examination. If this is not successful, the mass may be softened by warm water or saline lavage through a sigmoidoscope or rectal tube, or via a mineral oil enema. Rarely, surgical removal of the impaction is necessary. A combination of behavioral and medication therapy and maintenance management yields the most favorable outcomes in children with functional constipation and soiling.

DIVERTICULITIS

Diverticulosis refers to the presence of diverticula, or herniations of the intestinal mucosa and submucosa, commonly present in the sigmoid colon. More than half of patients older than 50 years have incidental colonic diverticula. *Diverticulitis* is the most frequent complication of diverticulosis, occurring in up to 20% of patients, and results from a microperforation of a diverticulum by the presence of inspissated fecal material that often becomes a phlegmon, or a pericolic or intra-abdominal abscess.

Symptoms

- Most people with colonic diverticula are asymptomatic; in contrast, abdominal pain is the presenting symptom in almost all patients with diverticulitis. Other presentations are rare except in older adults and diabetic patients in whom abdominal infections may sometimes present with minimal abdominal complaints and findings. Some may complain of chronic or intermittent LLQ abdominal pain. ++++
- RLQ pain + (in some Asian populations +++)
- Infrequent bowel movements or constipation +++
- Flatulence ++
- Dyspepsia ++
- Nausea ++
- Vomiting ++
- Constipation ++
- Diarrhea ++
- Dysuria +
- Urinary frequency +
- Symptoms may overlap with irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), colon cancer, ischemic colitis, bowel obstruction, and gynecologic and urologic disorders.

Signs

- LLQ tenderness +++++
- Fever ++++
- Leukocytosis ++++
- RLQ tenderness + (in some Asian populations +++)

Workup

- The American Society of Colon and Rectal Surgeons' parameters for the treatment of diverticulitis state that if a patient's clinical picture clearly suggests acute diverticulitis based on symptomatology, the diagnosis can be made on the basis of clinical criteria alone.
- The need for additional tests in a patient with suspected diverticulitis is determined by the severity of the presenting signs and symptoms and security of the diagnosis of diverticulitis.
- CT scan with oral, rectal, and intravenous (IV) contrast has become increasingly used as the initial imaging test for patients with suspected diverticulitis, particularly when moderate severity disease or abscess is anticipated. Due to the risks of extravasation of contrast from a potential colonic perforation in the patient with acute diverticulitis, barium enemas should be avoided in patients in whom perforation is a significant consideration.
- Criteria for the diagnosis of diverticulitis via water-soluble contrast enema include the presence of diverticula, mass effect, intramural mass, sinus tract, and extravasation of contrast.
- US may reveal bowel wall thickening, abscess, and rigid hyperechogenicity of the colon due to inflammation and may be helpful in female patients to exclude pelvic or gynecologic pathology.
- Criteria for the diagnosis of diverticulitis include colonic wall thickening, pericolic fat infiltration (“streaky” fat), pericolic or distant abscesses, and extraluminal air.
- CT is not sufficiently sensitive to differentiate cancer from diverticulitis and must be followed after acute treatment by contrast enema or endoscopy.
- Endoscopy is commonly avoided in the setting of acute diverticulitis because of the risk of perforating the inflamed colon, either with the instrument itself or by the insufflation of air.
- In situations in which the diagnosis of acute colonic diverticulitis is uncertain, limited flexible sigmoidoscopy with minimum insufflation of air may be performed to exclude other diagnoses.

Comments and Treatment Considerations

The decision of whether to proceed with inpatient or outpatient treatment of diverticulitis depends on the clinical judgment of the physician, the severity of the disease process, and the likelihood that the patient's condition will respond to outpatient therapy.

Patients who are able to tolerate a diet, who do not have systemic symptoms, and who do not have significant peritoneal signs may be treated on an outpatient basis with trimethoprim-sulfamethoxazole (TMP-SMX) or a fluoroquinolone plus metronidazole for 10 days. Conservative treatment results in resolution in 70% to 100% of cases.

A single dose of an IV antibiotic with coverage against gram-negative aerobes, either IV quinolone and metronidazole or oral TMP-SMX and metronidazole has been shown to be as effective as combination therapy in acute diverticulitis. A common regimen used is levofloxacin and metronidazole (Flagyl).

If the patient does not improve after several days, a colonic abscess should be suspected and diagnostic imaging should be considered.

After recovering from an initial episode of diverticulitis and when the inflammation has settled, the patient should be reevaluated. Appropriate examinations include a combination of flexible sigmoidoscopy and single- or double-contrast barium enema or colonoscopy.

Eventual resumption of a high-fiber diet is recommended after acute inflammation resolves; long-term fiber supplementation after the first episode of diverticulitis has been shown to prevent recurrence in more than 70% of patients followed up for more than 5 years.

Surgical treatment of diverticulitis, in the acute and chronic settings, has been successfully accomplished by laparoscopic and laparoscopic-assisted techniques. Primary colonic resection and anastomosis without a protective stoma has become the surgical treatment of choice for uncomplicated diverticulitis and may also be performed for patients with localized pericolic or pelvic abscesses.

Factors considered when deciding whether to proceed with resection include physiologic age of the patient; the number, severity, and interval of the attacks of diverticulitis; the rapidity and degree of response to medical therapy; and the persistence of symptoms after an acute attack of diverticulitis.

The risk of recurrent symptoms after an attack of diverticulitis ranges from 7% to 45%. With each recurrent episode, the patient is less likely to respond to medical therapy (70% chance of response to medical therapy after the first attack compared with a 6% chance after the third). Thus after two attacks of uncomplicated diverticulitis, resection is commonly recommended.

Free perforation of acute diverticulitis with fecal or purulent peritonitis is a surgical emergency that requires immediate resuscitation with IV fluids, broad-spectrum antibiotics, cardiovascular support (when indicated), and prompt operative therapy.

DYSPEPSIA

Dyspepsia (literally, "bad digestion") accounts for approximately 5% of all visits to family practitioners and is the most common reason for a referral to a gastroenterologist in the United States, comprising 20% to 40% of consultations. The lack of a standardized definition affects accurate prevalence data, given the challenge of clearly defining dyspepsia as either *functional* or *non-ulcer dyspepsia* (NUD) (approximately 60% of cases), or that caused by structural or biochemical disease (40% of cases). Regardless of cause, dyspepsia

has a profoundly negative effect on patients' health-related quality of life (HRQOL) and provides a significant economic burden.

Studies examining factors that drive a primary care provider's decision to refer a patient for a gastroenterologic evaluation of dyspepsia are lacking. As a consequence, many patients with organic dyspepsia due to gastroesophageal reflux disease (GERD), peptic ulcer disease (PUD), or even malignancy are most often managed empirically in primary care. Formal management strategies of uninvestigated dyspepsia have been designed to reduce the number of endoscopic procedures, and ultimately, direct cost and inconvenience to the patient (Fig. 1-1).

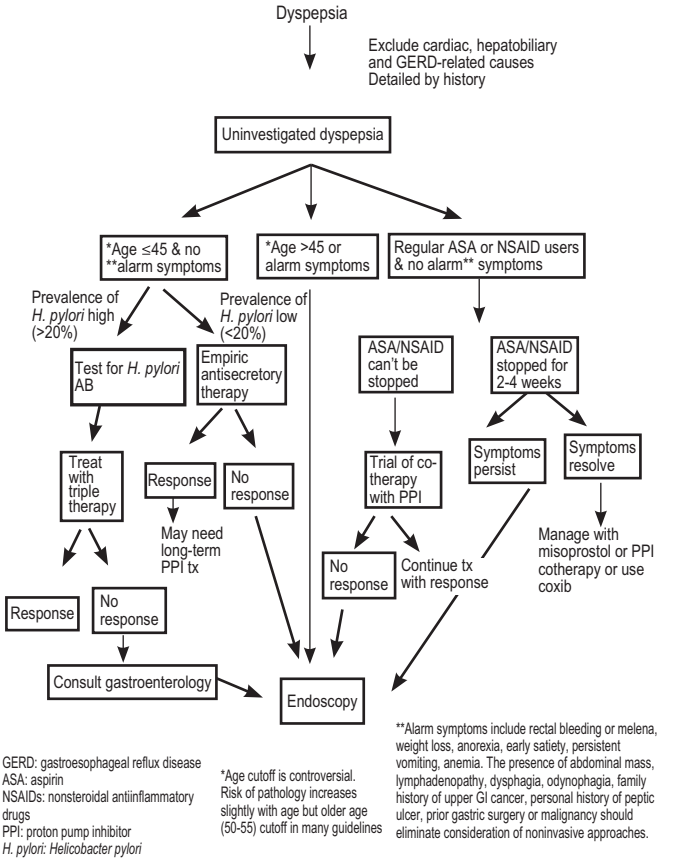

FIGURE 1–1 Evaluation of uninvestigated dyspepsia. *(Adapted from Saad R, Scheiman JM: Diagnosis and management of peptic ulcer disease,* Clin Fam Pract *6:569-587, 2004.)*

Symptoms

- Episodic or recurrent epigastric "aching," "gnawing," or "hunger-like" pain or discomfort arising from the proximal GI tract on an empty stomach commonly relieved by meals ++++
- Gastric ulcer pain often occurs 5 to 15 minutes after eating and remains until the stomach empties, which may be up to several hours in duration, whereas epigastric pain is otherwise absent during times of fasting. ++++
- Pain caused by duodenal ulcers is often relieved by eating, drinking milk, or taking antacids, and may return anywhere from 90 minutes to 4 hours after eating a meal. ++++
- Both classifications of ulcers may be associated with nausea and vomiting occurring anytime shortly after eating to several hours later. ++++
- Indigestion ++++
- Heartburn +++
- Bloating +++
- Nausea commonly occurs in the setting of a gastric outlet obstruction, due to either the scarring from ulcer healing or inflammation with impaired gastric emptying. ++
- Pain may radiate to the back (suggestive of a penetrating ulcer of the posterior duodenum that erodes the pancreas). ++
- Regurgitation ++
- Early satiety ++

Signs

- Physical examination is often unreliable ++++
- Rigidity of the abdomen and absent bowel sounds suggest perforation. ++++
- Occasional upper abdominal or epigastric tenderness and guarding +++
- Weight loss ++

Workup

- FOBT
- Upper endoscopy is the preferred diagnostic modality. Some clinicians may prefer barium radiographic studies due to their availability compared to the upper endoscopy.
- Long-term cost comparisons have been shown to favor endoscopy over other diagnostic modalities, and patients have been found to actually prefer endoscopy to that of barium radiographic studies in the diagnosis of PUD.
- Upper endoscopy provides the distinct advantage of permitting biopsies and/or brushings to identify the presence of underlying pathology, namely *Helicobacter pylori* and malignancy.

Comments and Treatment Considerations—*Helicobacter pylori*

Fecal-oral infection with *H. pylori* is a major risk factor for the development of PUD. Its prevalence and association with PUD is higher in populations where the standard of living is considered to be lower than that of the United States, especially in Africa and Central

America. Approximately 90% of patients worldwide with duodenal ulcers are infected with *H. pylori*, yet in the United States, its association with PUD ranges from 30% to 60%. The strongest evidence to support the role of *H. pylori* as an etiology of PUD is the elimination of ulcer recurrence when the infection has been successfully eradicated.

Patients younger than 45 years with dyspepsia and no alarm symptoms (see Fig. 1-1) should be tested for *H. pylori* infection and then given eradication therapy if positive ("test and treat"), whereas patients older than 45 years and those with alarm symptoms should have prompt endoscopy.

H. pylori–negative patients younger than 45 years of age and without alarm symptoms should be managed empirically for functional dyspepsia.

H. pylori–positive or *H. pylori*–negative patients who do not undergo endoscopy initially should do so if their symptoms persist. The effectiveness of this approach depends on the prevalence of *H. pylori* infection in patients with ulcers in the community because in some geographic areas prevalence may be too low to make this approach effective.

H. pylori testing via nonendoscopic methods includes a quantitative assay for serum immunoglobulin G (IgG) antibodies, the radiolabeled urea breath test, and the stool antigen test. The median sensitivity and specificity for serologic IgG tests are 92% and 83%, respectively. Some patients may have persistently positive IgG antibodies for months to years after eradication therapy, yielding a false-positive result during that period if retested.

The stool antigen test has been recommended by the European *Helicobacter pylori* Study Group as the preferred initial noninvasive diagnostic test; the urea breath test is the recommended standard to determine if *H. pylori* has been successfully eradicated.

Once *H. pylori* has been identified in the setting of PUD, treatment should be initiated regardless of whether there is a history of nonsteroidal antiinflammatory drug (NSAID) use, yet treatment of *H. pylori* alone is not adequate in preventing ulcer recurrence if NSAID therapy is continued or restarted.

Although most *H. pylori*–infected patients do not develop an ulcer, as many as 95% of patients with duodenal ulcers and 80% of those with gastric ulcers are infected. Patient education regarding the need for effective eradication therapy and to encourage adherence to the drug regimen is critical.

A meta-analysis of random controlled trials (RCTs) of *H. pylori* eradication for the treatment of duodenal ulcers found that one ulcer recurrence (evidenced on endoscopy) would be prevented for every 2.8 patients successfully treated. The best evidence-based recommendation for *H. pylori* eradication is for 14-day triple therapy with the use of a proton pump inhibitor (PPI), clarithromycin, and either amoxicillin or metronidazole, yielding eradication rates from 75% to 90%.

Although no studies demonstrate any difference among the available PPIs when used in the triple therapy regimens, the chosen

antibiotic has been shown to effect the eradication rates due to various antibiotic resistances. The current resistance rates of *H. pylori* in the United States are 33% for metronidazole, 11% for clarithromycin, and 0% for amoxicillin. The ideal *H. pylori* eradication regimen should reach an intention-to-treat cure rate of 80%.

Patients with documented PUD should have adequate follow-up because further diagnostic testing may be needed to ensure eradication of the *H. pylori* organism, particularly in the case of treatment failure or relapse. Because eradication therapy usually cures PUD, chronic acid suppression therapy should not be needed in most patients who have cleared the *H. pylori* infection and who are not taking NSAIDs.

Among primary care patients with a history of PUD taking chronic acid suppressive therapy, 78% of those treated for *H. pylori* were able to discontinue their therapy.

Comments and Treatment Considerations—NSAIDs

The use and overuse of these medications is the most common cause of PUD in *H. pylori*–negative patients, and up to 60% of unexplained cases of PUD are attributed to unrecognized NSAID use. Independent risk factors that augment the effect of *H. pylori* and/or NSAID-related PUD risk, and may promote ulcer complications include advancing age; a history of PUD or complicated ulcer disease with perforation, penetration, or gastric outlet obstruction; multiple NSAID use (including the concomitant use of low-dose aspirin and an NSAID); and concurrent warfarin or corticosteroid use.

Evidence suggests that smoking may augment the risk of PUD and ulcer complications by impairing gastric mucosal healing. Alcohol use may increase the risk of ulcer complications in NSAID users, but its overall effect in those patients without concomitant liver disease has not been clearly defined. There is no solid evidence to implicate dietary factors in the development of PUD.

It has been recognized that there is an increased familial incidence of PUD, most likely due to the familial clustering of *H. pylori*, and inherited genetic factors. NSAID and aspirin use is frequently associated with symptoms of dyspepsia, even in the absence of PUD. Empiric antisecretory therapy with PPIs is an attractive strategy that involves subjecting only those patients to upper endoscopy who fail to respond to a 4-week course of pharmacotherapy.

The approach to NSAID-related PUD can be divided into primary prevention, the promotion of ulcer healing, and the prevention of recurrence and its complications. The optimal management plan is the avoidance of NSAIDs in high-risk individuals (particularly in older adults), in patients with a history of PUD, and in patients taking corticosteroids and/or anticoagulants.

Management strategies for the primary prevention of NSAID-induced PUD include cotherapy with either a histamine type-2 receptor antagonist (H_2RA) or PPI, and the eradication of *H. pylori*, if present.

INFLAMMATORY BOWEL DISEASE

The incidence of ulcerative colitis (UC) and Crohn's disease is approximately 1.5 to 8 new cases per 100,000 persons per year in the United States. It is more commonly seen among whites and has no specific gender predominance, although some reviews have suggested a male predominance in Crohn's disease and a female predominance in UC. Most patients are diagnosed with IBD between the ages of 15 and 25, and there is a second peak of incidence between 55 and 65 years of age. Genetic factors have been implicated because first-degree relatives of patients with either form of IBD have been shown to have an almost 10% lifetime risk of developing disease, and they commonly present with a similar disease type and course as their affected family member. Environmental factors are also believed to be important in the pathogenesis of IBD. Although remarkably rare in Africa and Asia, the risk of IBD has been shown to increase when individuals migrate to a higher-risk region, such as the United States or Western Europe.

ULCERATIVE COLITIS

Symptoms

- Crampy lower abdominal pain +++++
- Mild to moderate diarrhea without constitutional symptoms ++++
- The more severe the illness, the greater the number of bowel movements, and the more likely constitutional symptoms such as fever, fatigue, dehydration, and weight loss also occur. ++++
- Can be intermittent with flare-ups and remission can occur without therapy ++
- A minority of patients with UC present with severe or fulminant pancolitis, ranging from an acute abdomen to toxic megacolon. ++

Signs

- Rectal bleeding +++++
- Hematochezia +++++
- Involves the mucosal layer of the sigmoid colon and rectum in the vast majority of cases, causing proctitis and proctosigmoiditis +++++
- When there is proximal spread, it tends to be continuous and symmetric, causing intestinal mucosal inflammation with edema and friability that is visualized from the rectum proximally. +++++
- Pancolitis is caused by inflammatory exudates producing a "backwash ileitis" by way of a patent ileocecal valve, and can cause small bowel involvement. ++++
- Chronic ulcerative colitis with cycles of flares and healing can produce scarring and shortening of the colon. ++++
- Weight loss ++++
- Anemia ++++

CROHN'S DISEASE

Symptoms

- In mild cases, or when only a few inches of the terminal ileum are involved, abdominal pain may be vague, the diarrhea intermittent, and weight loss absent. ++++
- In cases with more extensive small bowel and/or colonic involvement, the presentation often consists of significant abdominal pain (often in the RLQ) and frequent diarrhea. ++++
- Rectal involvement produces more urgent and frequent small, bloody stools as a result of an inflamed, nondistensible rectum. ++++
- Mild to moderate abdominal tenderness ++++
- Postprandial crampy pain can suggest transient small bowel obstruction from inflamed or fibrotic narrowed small bowel segments. +++
- Colonic involvement with Crohn's disease may present similar to that seen in UC, with predominantly bloody diarrhea. +++
- Anorexia +++
- Pallor due to anemia of blood loss of chronic disease +++
- Low-grade fever ++
- Tachycardia secondary to dehydration and diminished blood volume +
- Malnourishment (potential) +

Signs

- Mucosal abnormalities are discontinuous ("skip lesions"), asymmetric, and patchy, which accounts for obstruction, abscesses, and perianal fistulae. Lesions to other organs and skin can also be seen in Crohn's disease. +++++
- Most commonly found in the immunologically rich terminal ileum +++++, and involves the rectum in less than 50% of cases. ++
- Weight loss ++++
- Anemia ++++
- Perianal scarring or fistulae ++++
- May involve any part of the GI tract from the mouth to the anus, including the gallbladder and biliary tree, and involves the entire thickness of the bowel wall ++++
- Endoscopic appearance of "rake marks" or "cobblestone patterns" ++++
- Recurrent disease flares and healing of the disease can result in significant muscular hypertrophy and fibrosis of the intestinal wall that lead to small bowel strictures, upstream dilation of intestine and increased fistula formation, eventual bowel obstruction and the imminent need for surgical intervention. ++++
- Abdominal distention +++
- Rebound tenderness, absence of bowel sounds, and high fever may indicate toxic megacolon. +++
- Extraintestinal manifestations of Crohn's disease:
  - Joints—Arthritis, sacroiliitis, ankylosing spondylitis +++
  - Skin—Erythema nodosum, pyoderma gangrenosum ++

- Eyes—Conjunctivitis, iritis ++
- Liver—Fatty infiltration, chronic active hepatitis, primary sclerosing cholangitis, pericholangitis, bile duct carcinoma ++
- Kidneys—Pyelonephritis, renal stones ++
- Oral—Aphthous ulcers ++
- Amyloidosis +

Workup

- Patients presenting with diarrhea containing blood and/or mucus should undergo an appropriate workup including FOBT, fecal leukocytes and lactoferrin, stool cultures, and ova and parasite smears.
- In the vast majority of cases, diarrhea is caused by infections by viral and less commonly bacterial agents. For persistent or recurrent or particularly severe complaints with abdominal findings, a diagnosis of IBD should be considered, and a prompt referral to a gastroenterologist should be arranged.
- Flexible sigmoidoscopy and colonoscopy allow for direct visualization and biopsy of colonic mucosa. Endoscopic biopsy results consistent with nonspecific inflammation are not helpful.
- Patients with explained diarrhea and hematochezia should undergo colonoscopy to rule out cancer.
- Patients with UC and Crohn's disease have an increased risk of colorectal cancer and should be followed with routine surveillance colonoscopy.
- The finding of confluent erythematous rectal inflammation is most consistent with UC and infectious colitis.
- Pseudopolyp formations indicate chronic inflammatory colitis, whereas solitary aphthous ulcers, "rakelike" lesions, strictures, and rectal sparing are consistent with Crohn's disease.
- Colonoscopic evaluation should include ileal intubation and biopsies of both normal and abnormal mucosa. Extreme caution should be taken during colonoscopy given a high risk of iatrogenic perforation.
- Anal or perianal lesions, including sinus tracts, rectovaginal fistulae, and abscesses, is consistent with Crohn's disease but not with UC. The mucosa in a patient with Crohn's disease may appear cobblestoned or nodular. Loss of haustra, distortion of normal architecture, or both may be found.
- Laboratory values usually include an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), and decreased hemoglobin and serum albumin, giving an indication of the chronicity and severity of disease.
- An elevated alkaline phosphatase in a patient who has known UC always should raise the question of coexisting primary sclerosing cholangitis.

Comments and Treatment Considerations

Pharmacologic treatment of IBD is aimed at inducing remission and maintaining a symptom-free life, and is often directed after consultation with a gastroenterologist. Treatment of active flares with systemic corticosteroids has been the mainstay of remission-induction

therapy and produce remission rates of 70% in Crohn's disease versus 30% with placebo; similar results have been shown in the remission of UC.

Budesonide, a nonsystemic steroid used in an enema formulation, has been shown to be effective for the induction of remission in Crohn's disease and distal UC flares. Mild flares of UC are commonly treated with 5-aminosalicylic acid (5-ASA) derivatives such as sulfasalazine, yet RCTs have shown it is only marginally superior to placebo at controlling flares of Crohn's disease. 5-ASA and mesalamine can be used in enema or suppository formulation for patients with left-sided ulcerative colitis or proctitis; 5-ASA products are used infrequently for the induction of remission in Crohn's disease.

Azathioprine and its metabolite, 6-mercaptopurine, are slow-acting compounds proven to be effective for inducing remission in Crohn's disease. They are often added to systemic steroids to help induce and maintain remission and to ease steroid tapering. Patients using these medications should not be exposed to live vaccines and should receive high priority for annual influenza vaccines and Pneumovax.

Metronidazole and ciprofloxacin have been used for induction of remission in patients with active Crohn's disease. No evidence of benefit in induction of remission in UC has been demonstrated by using antibiotics.

Methotrexate is also effective for the induction of remission in Crohn's disease. Close monitoring of CBCs and serum transaminases is recommended, with monthly testing on initiation and with dosage changes. Pregnancy and exposure to live vaccines should be avoided.

Infliximab, an antitumor necrosis factor-α antibody, is remarkably effective in treating approximately 60% of steroid-resistant patients with Crohn's disease. It has significant side effect risks, including infusion reactions, worsening of heart failure, activation of latent tuberculosis, serum sickness, and invasive fungal infections.

In patients who are in remission and are not receiving maintenance therapy, 50% of those who have Crohn's disease will have a flare within 2 years and approximately 89% of patients who have UC will relapse within 1 year. The risk of undergoing exploratory or bowel resection surgery for complications of Crohn's disease is approximately 60% by 10 years from the diagnosis; this risk increases with an early age at diagnosis and previous surgery for Crohn's disease.

INTESTINAL ISCHEMIA

The spectrum of ischemic bowel disease is broad, and is best divided into acute and chronic mesenteric ischemia, and colonic ischemia. Management of each of these categories of ischemic injury requires its own unique strategy, largely based on descriptive studies and clinical experience summarized in the American Gastroenterological Association guidelines for intestinal ischemia because evidence based on RCTs is lacking.

Symptoms

- Acute mesenteric ischemia—Severe abdominal pain particularly in older adults and those with vascular disease whose clinical picture does not suggest another abdominal diagnosis (e.g., cholecystitis or diverticulitis) ++++
- Chronic mesenteric ischemia ("intestinal angina")—Postprandial abdominal pain and marked weight loss resulting from repeated transient episodes of inadequate intestinal blood flow provoked by the increased metabolic demands associated with digestion ("intestinal angina") ++++
- Colonic ischemia—Mild to moderate abdominal pain, diarrhea, or lower GI bleeding with minimal to moderate abdominal tenderness ++++
- Hematochezia ++++

Signs

- In acute ischemia of the bowel, pain may be severe with abdominal findings coming late at the time of bowel infarction.
- Diffuse or localized abdominal tenderness with guarding and/or rebound tenderness (a late finding) +++++
- Decreased bowel sounds ++++
- Weight loss +++
- Abdominal bruit (nondiagnostic) ++

Workup

- Acute and chronic mesenteric ischemia—Emergent surgical consultation, mesenteric angiography, CT or magnetic resonance imaging (MRI)
- Colonic ischemia—Colonoscopy or barium enema; mesenteric angiography is useful only if the ascending colon is affected

Comments and Treatment Considerations

Acute mesenteric ischemia results from either arterial or venous thrombi, or vasoconstriction secondary to decreased blood flow. Correctly diagnosing this condition prior to intestinal infarction is the single most important factor in minimizing poor outcomes, as reported mortality rates range from 59% to 93%. Patients at risk for acute mesenteric ischemia, most notably those with atherosclerotic disease or procoagulant conditions, who have severe abdominal pain should be promptly evaluated. These patients must be identified early in the clinical course of the disease and treated aggressively if survival is to be improved.

Suspected patients should undergo emergent surgical exploration or mesenteric angiography if another cause for the pain cannot be discovered via abdominal plain film radiography or CT. Vasodilators are widely used in the treatment of nonocclusive mesenteric ischemia, and are strongly suggested for occlusive disease of the superior mesenteric artery. Anticoagulants and thrombolytics have also been used, but rigorous studies comparing their outcomes to that of other modalities are lacking. In patients who develop acute ischemic colitis, expert opinion states that systemic corticosteroids should be avoided.

In patients with chronic mesenteric ischemia, mesenteric angiography commonly demonstrates partial or complete occlusion of at least two of the three major splanchnic vessels, yet these abnormalities alone are not sufficient to diagnose chronic mesenteric ischemia.

Treatment is either surgical or by percutaneous transluminal mesenteric angioplasty with or without stenting. Experience with angiographic treatment modalities is limited, and at present these modalities are best reserved for patients at a high risk for surgical revascularization. High-resolution CT and MRI have been proposed for use in diagnosing chronic mesenteric ischemia, but have not been proven sufficiently sensitive or specific to become a gold standard.

Colonic ischemia is the most common form of intestinal ischemia. Although most cases have no definable cause, it is often associated with a spectrum of disorders including transient colitis, chronic colitis, stricture, gangrene, and fulminant universal colitis. Diagnosis is made via colonoscopy or barium enema in an individual with a typical history.

Broad-spectrum antibiotics are customarily used in treatment, despite an absence of solid clinical evidence supporting their benefit. Although most cases of colonic ischemia have an excellent prognosis and resolve spontaneously, surgery may be required acutely, subacutely, or electively in chronic cases. Surgery is indicated acutely for those patients with peritoneal signs, massive GI bleeding, or fulminant colitis; subacutely for those who do not improve after 2 to 3 weeks or who develop recurrent sepsis; and electively in cases of symptomatic ischemic stricture or chronic colitis.

IRRITABLE BOWEL SYNDROME

IBS is one of the most common GI conditions encountered in family practices with a prevalence ranging from 14% to 24% in women and from 5% to 19% in men in the United States and England. The syndrome commonly appears in the late 20s, although it may present in teenagers and in patients as old as age 45; patients older than 45 years with suspected IBS should be evaluated for organic disease. Although the diagnosis of IBS may be a challenging one to make, it is clear that symptoms suggestive of IBS are common, and less than 25% of symptomatic patients seek medical advice for their symptoms. IBS is responsible for approximately 2.4 to 3.5 million physician visits per year in the United States, and represents 12% of primary care visits and 28% of referrals to gastroenterologists.

Studies have indicated that the HRQOL in patients in the United States with IBS is worse than that of patients suffering from clinical depression. Patients who have IBS are much more likely to exhibit health care–seeking behaviors that are related to GI and non-GI complaints. Consultations for non-GI problems are four times more common in this population compared with patients who do not have IBS.

Psychosocial stressors likely exacerbate symptoms in patients with functional GI disorders. Anxiety disorders, somatoform disorders, and a history of physical and/or sexual abuse have been identified in 42% to 61% of patients with IBS who have been referred to gastroenterologists. Patients with IBS have a hypersensitivity to bowel stimulation with stress and lumen exposures to food and bacteria as known exacerbating factors. In addition, autonomic nervous system dysfunction occurs, which has been shown to alter visceral perception and has reproducibly been shown to accelerate large bowel motility and delay gastric emptying. Overlap syndromes with fibromyalgia and interstitial cystitis have led to multidisciplinary approaches to the management of IBS with an emphasis on behavior therapies to improve HRQOL.

Symptoms

- Abdominal pain +++++
- Cramping +++++
- Bloating +++++
- Constipation or diarrhea, or both +++++
- Anxiety ++++
- Depression ++++
- Mucus in stools +++

Signs

- Physical examination is often nonspecific, and may demonstrate a normal abdominal examination, a diffusely tender abdomen, or a focally tender abdomen. ++++
- Voluntary guarding ++++

Workup

- The Rome II criteria are the most widely accepted classification for establishing a diagnosis of IBS (Table 1-1).
- Multiple diagnostic screening tests have been recommended including a CBC, ESR, serum chemistries, thyroid function tests,

Table 1-1. Rome II Criteria for Diagnosis of Irritable Bowel Syndrome

- Abdominal pain or discomfort for at least 12 weeks, although not necessarily contiguous, during the last 12 months
- At least two out of three of the following features:
  - Relief with defecation
  - Onset associated with change in form of the stool
  - Onset with change in the frequency of bowel movements
- Additional supporting features include:
  - Fewer than three bowel movements per week
  - More than three bowel movements per day
  - Hard or lumpy stool

Adapted from Cash BD, Chey WD: Irritable bowel syndrome: a systematic review, *Clin Fam Pract* 6:647-669, 2004.

stool cultures including ova and parasites, FOBT, colonoscopy, and hydrogen breath testing, specifically to rule out other causes of disease.
- Despite these recommendations, diagnostic testing should depend on the pretest probability of organic disease.
- The pretest probability of IBS depends on the presence or absence of alarm symptoms, including hematochezia, fevers, weight loss greater than 10 pounds, chronic severe diarrhea, and family history of colon cancer.
- In the absence of alarm symptoms, diagnostic testing should be limited. Studies have suggested that accurate diagnosis of IBS is imperative (although challenging), and that protracted negative workups may negatively affect symptomatology and outcomes.
- Diagnostic testing may be performed to reassure the clinician as well as the patient, yet the value of reassurance from negative diagnostic testing has never been examined.
- The differential diagnosis of IBS includes IBD, lactose intolerance, acute gastroenteritis, celiac disease, small intestinal bacterial overgrowth, colorectal cancer, and motility-altering metabolic disturbances (e.g., hypo- or hyperthyroidism).

Comments and Treatment Considerations

There is no single evidence-based consistently successful therapeutic approach for patients with IBS. Because it is largely a chronic condition, the goals of therapy should focus on patient reassurance, education about the natural course of the syndrome, and global symptomatic improvement, rather than on disease cure. This is best achieved through a well-developed patient-physician relationship with a clear delineation of realistic goals and expectations. Treatment for symptoms related to IBS is indicated when the patient and physician believe there has been a decrement in HRQOL.

Alosetron, a 5-HT3 antagonist, is indicated for women with diarrhea-predominant IBS, and has also been shown to be more effective than placebo in RCTs. Due to reports of ischemic colitis, the use of alosetron has been limited to physicians participating in the manufacturer's risk management program.

Treatment of diarrhea-predominant IBS can be achieved with the use of loperamide; however, no advantage over placebo for global IBS symptoms has been reported. To date, all other classes of medications used in the management of IBS have a more limited effect on the global symptoms of IBS. The tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs), and peppermint oil have been shown to reduce abdominal pain.

Treatment of constipation-predominant IBS can be achieved with fiber-bulking agents. Cognitive behavioral therapy, interpersonal psychotherapy, group therapy, biofeedback, and hypnosis have been shown to improve individual aspects of diarrhea-predominant IBS. Alternative medicine techniques, including acupuncture, probiotic therapy, and Chinese herbal medicine, are becoming increasingly

popular in the treatment of GI disorders, and have been shown to have some limited symptomatic improvement in selected cases of IBS.

PANCREATITIS

Mild acute pancreatitis, accounting for almost 80% of all cases, is characterized by parenchymal interstitial edema of the pancreas, and no worse than minimal distal organ dysfunction. Recovery is usually rapid (measured in days) and any distal organs affected by the acute event quickly return to their baseline function. In severe acute pancreatitis, parenchymal and fat necrosis ensues, as well as profound multisystem organ failure, infection, and life-threatening hemodynamic instability. Many patients fall somewhere between these categories.

The causes of acute pancreatitis are diverse and demonstrate changing trends over time and variation by geography. Gallstones, biliary sludge, and microlithiasis are recognized as the proximate cause in well over half of reported cases in several studies from around the world. Ethyl alcohol ingestion is the second most commonly reported cause of acute pancreatitis and accounts for up to 30% of cases. The remaining causes of acute pancreatitis account for less than 15% of total cases by most accounts, including hypertriglyceridemia, trauma, medications, endoscopic retrograde cholangiopancreatography (ERCP), neoplasms, perforated peptic ulcer disease, viral infection, and idiopathic causes. Many of the cases of idiopathic pancreatitis may be due to unrecognized microlithiasis.

Symptoms

- Gnawing epigastric abdominal pain radiating to the back that is commonly constant and can last from hours to days, but most often has been present for more than 24 hours +++++
- Substernal pain, generalized to the left upper quadrant (LUQ) or right upper quadrant (RUQ), or even the lower abdomen, worsened by food or alcohol, and may be precipitated by binge drinking ++++
- Abdominal distention ++++
- Nausea ++++
- Vomiting +++

Signs

- Decreased bowel sounds on auscultation +++
- Small bowel ileus, secondary to diffusion of inflammatory fluid around the pancreas ++
- Low-grade fever of 37.8° to 38.9° C (100° to 102° F) may be present; a temperature in excess of 38.9° C (102° F) suggests another diagnosis or complication ++
- Hypovolemia ++
- Cullen's sign, a periumbilical bluish discoloration +
- Grey Turner's sign, a bluish discoloration of the flanks +
  - *Neither of these signs are specific for pancreatitis and are rarely seen.*

Workup

- Lipase is the serum marker of choice with high sensitivity and specificity. However, lipase level does not predict the severity or course of disease.
- The sensitivity of pancreatic amylase for the diagnosis of acute pancreatitis decreases to less than 30% between the second and fourth day after the onset of the acute episode.
- Amylase levels may be elevated in a variety of nonpancreatic conditions; a small bowel obstruction is the most relevant of these when abdominal pain is of an unclear etiology.
- The clearance of pancreatic amylase is diminished with a decline in renal function, thus may cloud the clinical picture even further.
- By contrast, an elevated serum lipase level can be detected as distant as 14 days after the acute event and has a sensitivity of greater than 90% for acute pancreatitis.
- Early prognostic factors that can be measured and indicate severity of disease include Ranson's criteria and more recently the Acute Physiology and Chronic Health Evaluation II (APACHE II) score (most commonly used).
- One factor considered to be significant in the management of patients with acute pancreatitis is intravascular volume status.
- The hematocrit may be high as a secondary effect of hypovolemia secondary to third spacing of fluids. In many circumstances, patients may be as many as 6 L intravascularly depleted. Volume resuscitation during the first 24 hours is extremely important and may minimize or even prevent pancreatic necrosis.
- If the bilirubin, liver transaminases, and alkaline phosphatase rise, a common bile duct (CBD) stone may exist. Similar lab abnormalities may occur in patients with chronic pancreatitis and a bile duct stricture. This possibility could be further explored with abdominal US and possibly ERCP, which can be both diagnostic and therapeutic.
- Hypocalcemia, hypoalbuminemia, hyperglycemia, and leukocytosis in the range of 15,000 to 20,000 WBC/mL are frequently found. Leukocytosis with more than 20,000 WBC/mL suggests a more severe disease.
- Because respiratory distress syndrome may ensue, chest radiographs and arterial blood gases should be considered. In severe cases, renal failure may appear despite adequate fluid intake, thus urinary output should be closely monitored,
- Contrast-enhanced CT (CECT) is the most extensively studied modality for the confirmation of acute pancreatitis and provides the highest level of sensitivity and specificity among existing imaging technologies. Limitations include overlying gas (often seen with pancreatitis-related ileus), excessive abdominal fat, and distortions of the skin from scarring that can make visualization of the underlying organs less reliable.
- US may be added to CT scan in patients who have suspected acute pancreatitis in the uncommon situation in which the biliary system is not well visualized.

- When the pancreas is visualized in the setting of acute disease, tissue abnormalities are detected in 90% of those studied.
- MRI and magnetic resonance cholangiopancreatography (MRCP) are two newer modalities that have specific yet limited roles in diagnosing acute pancreatitis. In contrast to CT imaging, MRI is more likely to uncover mild disease but requires longer scanning times that necessitate breath-holding for often uncomfortable periods of time within cramped spaces. This modality is of particular use in patients who have allergic reactions to iodinated IV contrast, impaired renal function, or are pregnant and in patients who should not be exposed to ionizing radiation. It is superior to conventional CT imaging at detecting gallstone disease and detailing anatomic anomalies, although experience in its use in the setting of acute pancreatitis is still limited. MRI should be reserved for individuals in whom CT scanning is contraindicated and when mild acute pancreatitis is suspected.

Comments and Treatment Considerations

Medical therapy of acute pancreatitis is primarily supportive, with the major objective being hemodynamic stabilization. Nutritional status and maintenance should be considered early in the course of acute pancreatitis to minimize morbidity and mortality risk.

Most patients are kept NPO until their abdominal pain subsides and appetite returns. After several days, commencement of total parenteral nutrition (TPN) or enteral feeding through a nasojejunal tube (still somewhat controversial) in the absence of a paralytic ileus should be considered. Pain management in the hospital setting is best achieved using morphine derivatives.

CARDIOVASCULAR

ABDOMINAL AORTIC ANEURYSM

The most common etiology of abdominal aortic aneurysm (AAA) is atherosclerosis. Additional etiologies include genetic diseases (e.g., Ehlers-Danlos syndrome), trauma, cystic medial necrosis (e.g., Marfan's syndrome), arteritis, inflammatory conditions, mycosis, and infection (e.g., syphilis). White males have the highest incidence of AAA; males are affected up to seven times more often than females. More than 75% of patients with AAA are older than age 60.

Symptoms

- Most aneurysms develop slowly over many years and are asymptomatic. ++++
- If an aneurysm expands rapidly or ruptures, or if blood dissects along the wall of the aorta, symptoms may develop suddenly and include:
  - Pulsating sensation in the abdomen ++++
  - Severe, sudden, and persistent abdominal pain that may radiate to groin, back, flank, or legs ++++
  - Abdominal rigidity ++++

- Anxiety ++++
- Nausea +++
- Vomiting +++

Signs

- In incidental cases, especially in thin individuals, a pulsatile mass may be palpated in the midline of the abdomen, yet sensitivity for detection is low when the AAA is less than 5 cm. +++
- In cases of rupture or dissection, cardiogenic shock may occur exhibited by:
  - Tachycardia +++++
  - Hypotension +++++
  - Clammy skin ++++
  - Pallor ++++
  - Venous thrombosis from iliocaval venous compression ++
  - Discoloration and pain of the feet with distal embolization of the thrombus within the aneurysm. ++

Workup

- Abdominal US is nearly 100% sensitive and specific in identifying an AAA and estimating its size. It is not very accurate in estimating the proximal extension to the renal or iliac arteries
- CT is recommended for preoperative AAA imaging and estimating size. The rate of false negatives is extremely low, and the CT scan can localize the proximal extent, detect the integrity of the wall, and rule out rupture.
- Angiography provides detailed arterial anatomy and can localize the aneurysm relative to the renal and visceral arteries.

Comments and Treatment Considerations

In patients with the clinical triad of abdominal and/or back pain, a pulsatile abdominal mass, and hypotension, immediate surgical evaluation is indicated regardless. In patients with AAAs, blood pressure and fasting serum lipid values should be monitored and controlled as recommended for patients with atherosclerotic disease. Patients with aneurysms or a family history of aneurysms should be advised to stop smoking and be offered smoking cessation interventions.

In most cases, according to Lederle (Lederle, 2003), patients with infrarenal or juxtarenal AAAs measuring 5.5 cm or larger should undergo repair to eliminate the risk of rupture. In most cases, patients with infrarenal or juxtarenal AAAs measuring 4.0 to 5.4 cm in diameter should be monitored by US or CT every 6 to 12 months to detect expansion. In patients with AAAs smaller than 4.0 cm in diameter, monitoring by US examination every 2 to 3 years is reasonable.

Surgical intervention is not recommended for asymptomatic infrarenal or juxtarenal AAAs if they measure less than 5.0 cm in diameter in men or less than 4.5 cm in diameter in women. Surgical repair can be beneficial in patients with infrarenal or juxtarenal AAAs 5.0 to 5.4 cm in diameter. Surgical repair is indicated in patients with suprarenal or type IV thoracoabdominal aortic aneurysms larger than 5.5 to 6.0 cm.

Some screening regimens suggest that men 65 years of age or older who are either the siblings or offspring of patients with AAAs should undergo physical examination and US screening for detection of AAAs, and that men who are 65 to 75 years of age who have ever smoked should undergo a physical examination and baseline US screening for detection of AAAs.

Perioperative administration of β-adrenergic blocking agents, in the absence of contraindications may reduce the risk of adverse cardiac events and mortality in patients with coronary artery disease undergoing surgical repair of atherosclerotic aortic aneurysms and their administration should be considered.

Beta-blockers should be considered to reduce the rate of aneurysm expansion in patients with aortic aneurysms. Periodic long-term surveillance imaging should be performed to monitor for an endoleak, to document shrinkage or stability of the excluded aneurysm sac, and to determine the need for further intervention in patients who have undergone endovascular repair of infrarenal aortic and/or iliac aneurysms.

Endovascular repair of infrarenal aortic and/or common iliac aneurysms is reasonable in patients at high risk of complications from open operations because of cardiopulmonary or other associated diseases. Endovascular repair of infrarenal aortic and/or common iliac aneurysms may be considered in patients at low or average surgical risk.

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